Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS? ### Status: Signing onto Dialog ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Status: Login successfulWelcome to DIALOG

Dialog level 05.05.00D

Last logoff: 08jun05 14:19:29
Logon file405 13jul05 10:41:15
              *** ANNOUNCEMENT ***
                      ***
--UPDATED: Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                      ***
NEW FILES RELEASED
***Aluminium Industry Abstracts (File 33)
***Ceramic Abstracts/World Ceramic Abstracts (File 335)
***CSA Life Sciences Abstracts (File 24)
***Corrosion Abstracts (File 46)
***Materials Business File (File 269)
***Engineered Materials Abstracts (File 293)
***CSA Aerospace & High Technology Database (File 108)
***CSA Technology Research Database (File 23)
***METADEX(r) (File 32)
***FDAnews (File 182)
***German Patents Fulltext (File 324)
                      ***
RESUMED UPDATING
***Canadian Business and Current Affairs (262)
***CorpTech (559)
                      ***
Chemical Structure Searching now available in Prous Science Drugs
of the Future (F453), IMS R&D Focus (F445), Beilstein Facts (F390),
and Derwent Chemistry Resource (F355).
                      ***
    >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
    >>>   of new databases, price changes, etc.   <<<
                      ****
KWIC is set to 50.
HILIGHT set on as '*'
PICKS is set ON as an alias for
5,159,143,358,340,344,348,447,73,155,349,266,10,34,434,42,43,50,65,71,91,94,14
4,198,304,370,467,444,357,156,157.
* * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.7.9 term=ASCII
                    *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
   1. Announcements (new files, reloads, etc.)
   2. Database, Rates, & Command Descriptions
   3. Help in Choosing Databases for Your Topic
   4. Customer Services (telephone assistance, training, seminars, etc.)
   5. Product Descriptions

Connections:
  6. DIALOG(R) Document Delivery
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      /H = Help   /L = Logoff   /NOMENU = Command Mode

Enter an option number to view information or to connect to an online
 service. Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
?
Terminal set to DLINK
                    *** DIALOG HOMEBASE(SM) Main Menu ***

  Information:
    1. Announcements (new files, reloads, etc.)
    2. Database, Rates, & Command Descriptions
    3. Help in Choosing Databases for Your Topic
    4. Customer Services (telephone assistance, training, seminars, etc.)
    5. Product Descriptions

  Connections:
    6. DIALOG(R) Document Delivery
    7. Data Star(R)

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      /H = Help   /L = Logoff   /NOMENU = Command Mode

Enter an option number to view information or to connect to an online
 service. Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? B picks

>>>   43 does not exist
>>>1 of the specified files is not available
       13jul05 10:41:24 User243038 Session D128.1
               $0.00   0.214 DialUnits FileHomeBase
     $0.00 Estimated cost FileHomeBase
     $0.03 TELNET
     $0.03 Estimated cost this search
     $0.03 Estimated total session cost   0.214 DialUnits

SYSTEM:OS - DIALOG OneSearch
  File   5:Biosis Previews(R) 1969-2005/Jul W1
         (c) 2005 BIOSIS
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog Corporation
*File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.

  File 143:Biol. & Agric. Index 1983-2005/Jun
         (c) 2005 The HW Wilson Co
  File 358:Current BioTech Abs 1983-2005/Jun
         (c) 2005 DECHEMA

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File 340:CLAIMS(R)/US Patent 1950-05/Jul 07
        (c) 2005 IFI/CLAIMS(R)
File 344:Chinese Patents Abs Aug 1985-2005/May
        (c) 2005 European Patent Office
File 348:EUROPEAN PATENTS 1978-2005/Jun W04
        (c) 2005 European Patent Office
File 447:IMS Patent Focus 2005/Jun
        (c) 2005 IMS Health & Affiliates
File  73:EMBASE 1974-2005/Jul 12
        (c) 2005 Elsevier Science B.V.
File 155:MEDLINE(R) 1951-2005/Jul W2
        (c) format only 2005 The Dialog Corp.
File 349:PCT FULLTEXT 1979-2005/UB=20050707,UT=20050630
        (c) 2005 WIPO/Univentio
File 266:FEDRIP 2005/Jun
        Comp & dist by NTIS, Intl Copyright All Rights Res
File  10:AGRICOLA 70-2005/Jul
        (c) format only 2005 The Dialog Corporation
File  34:SciSearch(R) Cited Ref Sci 1990-2005/Jul W1
        (c) 2005 Inst for Sci Info
File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
        (c) 1998 Inst for Sci Info
File  42:Pharmaceuticl News Idx 1974-2005/Jun W4
        (c)2005 ProQuest Info&Learning
File  50:CAB Abstracts 1972-2005/Jun
        (c) 2005 CAB International
File  65:Inside Conferences 1993-2005/Jul W2
        (c) 2005 BLDSC all rts. reserv.
File  71:ELSEVIER BIOBASE 1994-2005/Jul W1
        (c) 2005  Elsevier Science B.V.
File  91:MANTIS(TM) 1880-2005/Jun
        2001 (c) Action Potential
File  94:JICST-EPlus 1985-2005/May W4
        (c)2005 Japan Science and Tech Corp(JST)
File 144:Pascal 1973-2005/Jul W1
        (c) 2005 INIST/CNRS
File 198:Health Devices Alerts(R) 1977-2005/Jul W1
        (c) 2005 ECRI-nonprft agncy
File 304:The Merck Index Online(SM) 200(c) 2005 Merck & Co. Inc.
        5/S1
```
*File 304: File is now current to the 13th edition of The Merck Index
```
File 370:Science 1996-1999/Jul W3
        (c) 1999 AAAS
```
*File 370: This file is closed (no updates). Use File 47 for more current
information.
```
File 467:ExtraMED(tm) 2000/Dec
        (c) 2001 Informania Ltd.
```
*File 467: F467 no longer updates; see Help News467.   7.
```
File 444:New England Journal of Med. 1985-2005/Jun W4
        (c) 2005 Mass. Med. Soc.
File 357:Derwent Biotech Res. _1982-2005/Jul W2
        (c) 2005 Thomson Derwent & ISI
File 156:ToxFile 1965-2005/Jul W2
        (c) format only 2005 The Dialog Corporation
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*File 156: ToxFile has been reloaded with the 2005 MeSH.
Please see HELP NEWS 156 for details.
```
File 157:BIOSIS Toxicology (c) 2004 BIOSIS

        Set  Items  Description
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  ---   -----  -----------
? s level of transcription
     S1      1  LEVEL OF TRANSCRIPTION
? t s1
```

1/2/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2005 BIOSIS. All rts. reserv.

0014120784   BIOSIS NO.: 200300079503
Mitogen activated protein kinase phosphatases-1 and -2 are differentially
  regulated at the level of transcription in cultured rat cardiac
  ventricular myocytes.
AUTHOR: Fuller Stephen J (Reprint); Finn Stephen G (Reprint)
AUTHOR ADDRESS: NHLI London, London, UK**UK
JOURNAL: Circulation 106 (19 Supplement): pII-205 November 5, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions Chicago, IL, USA
November 17-20, 2002; 20021117
SPONSOR: American Heart Association
ISSN: 0009-7322 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English
REGISTRY NUMBERS: 306298-47-5: mitogen-activated protein kinase phosphatase
    1; 306748-07-2: mitogen-activated protein kinase phosphatase 2;
    16561-29-8: 12-O-tetradecanoyl phorbol 13-acetate; 50-76-0: actinomycin
    D
DESCRIPTORS:
  MAJOR CONCEPTS: Cardiovascular System--Transport and Circulation; Cell
    Biology; Enzymology--Biochemistry and Molecular Biophysics; Molecular
    Genetics--Biochemistry and Molecular Biophysics
  BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata,
    Animalia
  ORGANISMS: rat (Muridae)--neonate
  ORGANISMS: PARTS ETC: ventricular myocyte--circulatory system, *level of
    transcription*
  COMMON TAXONOMIC TERMS: Animals; Chordates; Mammals; Nonhuman Vertebrates
    ; Nonhuman Mammals; Rodents; Vertebrates
  CHEMICALS & BIOCHEMICALS: mitogen-activated protein kinase phosphatase
    1; mitogen-activated protein kinase phosphatase 2; 12-O-tetradecanoyl
    phorbol 13-acetate; actinomycin D
  GENE NAME: rat c-Jun gene (Muridae); rat c-Fos gene (Muridae)
  METHODS & EQUIPMENT: Western blot--genetic techniques, laboratory
    techniques
CONCEPT CODES:
  00520 General biology - Symposia, transactions and proceedings
  02502 Cytology - General
  02506 Cytology - Animal
  03502 Genetics - General
  03506 Genetics - Animal
  10060 Biochemistry studies - General
  10064 Biochemistry studies - Proteins, peptides and amino acids
  10802 Enzymes - General and comparative studies: coenzymes
  14504 Cardiovascular system - Physiology and biochemistry
BIOSYSTEMATIC CODES:
  86375 Muridae
? t TGF beta transcription
>>>'TGF' not recognized as set or accession number

? t transforming growth factor beta transcription
>>>'TRANSFORMING' not recognized as set or accession number
? s CtBP
        S2   1292   CTBP
? s s2 and Smad
             1292   S2
            12514   SMAD
        S3   53   S2 AND SMAD
? rd
>>>Duplicate detection is not supported for File 340.
>>>Duplicate detection is not supported for File 344.
>>>Duplicate detection is not supported for File 348.
>>>Duplicate detection is not supported for File 447.
>>>Duplicate detection is not supported for File 349.
>>>Duplicate detection is not supported for File 198.
>>>Duplicate detection is not supported for File 304.

>>>Records from unsupported files will be retained in the RD set.
...examined 50 records   (50)
...completed examining records
        S4   19   RD (unique items)
? t s4/all
>>>'ALL' not allowed as format type
? t s4/t/5
>>>'T' not a valid format name
? t s4/3

 4/3/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2005 BIOSIS. All rts. reserv.

0014936627   BIOSIS NO.: 200400307384
Proteins associated with type II bone morphogenetic protein receptor
  (BMPR-II) and identified by two-dimensional gel electrophoresis and mass
  spectrometry
AUTHOR: Hassel Sylke; Eichner Annegret; Yakymovych Mariya; Hellman Ulf;
  Knaus Petra; Souchelnytskyi Serhiy (Reprint)
AUTHOR ADDRESS: BMC, Ludwig Inst Canc Res, Box 595,Husargatan 3, SE-75124,
  Uppsala, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: serhiy.souchelnytskyi@licr.uu.se
JOURNAL: Proteomics 4 (5): p1346-1358 May 2004 2004
MEDIUM: print
ISSN: 1615-9853 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s4/all/5
>>>'ALL' not allowed as format type
? t s4/ab/5
>>>No matching display code(s) found in file(s): 65, 198, 304, 447

 4/AB/5   (Item 5 from file: 5)
DIALOG(R)File   5:(c) 2005 BIOSIS. All rts. reserv.

ABSTRACT: Although Evi-1 is thought to promote growth or block
  differentiation in some cell types, its biological functions have not
  been elucidated. To explore the mechanisms underlying Evi-1-induced
  oncogenesis, we investigated whether Evi-1 affects the signaling of
  transforming growth factor beta (TGF-beta), which inhibits proliferation

of a wide range of cell types and is one of the most studied growth
regulatory factors. We demonstrated that Evi-1 represses TGF-beta
signaling and antagonizes its growth-inhibitory effects. Two separate
regions of Evi-1 are responsible for this repression, one of which is the
first zinc-finger domain. Through this domain, Evi-1 physically interacts
with Smad3, an intracellular mediator of TGF-beta signaling, thereby
suppressing the transcriptional activity of Smad3. These results define a
novel function of Evi-1 as a repressor of signaling components of
TGF-beta. We also demonstrated that Evi-1 represses *Smad*-induced
transcriptional activation by recruiting *CtBP* as a corepressor. Evi-1
associates with CtBP1 through one of the *CtBP*-binding consensus motifs
within the region from amino acid 544 to 607, and this association is
required for the efficient inhibition of TGF-beta signaling. A specific
histone deacetylase (HDAc) inhibitor, trichostatin A (TSA), alleviates
Evi-1-mediated repression of TGF-beta signaling, suggesting that HDAc is
involved in transcriptional repression by Evi-1. This identifies a novel
function of Evi-1 as a member of corepressor complexes and suggests that
aberrant recruitment of corepressors is one of the mechanisms involved in
Evi-1-induced leukemogenesis. These results indicate that specific HDAc
inhibitors may be useful in the treatment of Evi-1-induced neoplastic
tumors, including myeloid leukemias.
? t s4/all/all
>>>'ALL' not allowed as format type
? t s4/3,ab
>>>No matching display code(s) found in file(s): 65, 198, 304, 447

4/3,AB/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2005 BIOSIS. All rts. reserv.

0014936627   BIOSIS NO.: 200400307384
**Proteins associated with type II bone morphogenetic protein receptor
(BMPR-II) and identified by two-dimensional gel electrophoresis and mass
spectrometry**
AUTHOR: Hassel Sylke; Eichner Annegret; Yakymovych Mariya; Hellman Ulf;
Knaus Petra; Souchelnytskyi Serhiy (Reprint)
AUTHOR ADDRESS: BMC, Ludwig Inst Canc Res, Box 595,Husargatan 3, SE-75124,
Uppsala, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: serhiy.souchelnytskyi@licr.uu.se
JOURNAL: Proteomics 4 (5): p1346-1358 May 2004 2004
MEDIUM: print
ISSN: 1615-9853 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Bone morphogenetic proteins (BMP) are polypeptide growth factors
that regulate cell differentiation and proliferation. BMPs bind to type I
and type II serine/threonine kinase receptors to initiate intracellular
signalling. BMPR-II is the type II receptor, its mutations lead to
hereditary pulmonary hypertension, and knockout of Bmpr-II results in
early embryonic lethality. To identify novel interacting proteins and
explore signalling pathways that can be initiated by BMPR-II, we
performed glutathione-S-transferase (GST) pull-down assays with BMPR-II
protein constructs fused to GST and extracts of mouse myoblast C2C12
cells. We generated three constructs which contain different parts of the
cytoplasmic region of BMPR-II: full-length cytoplasmic part of BMPR-II,
only the kinase domain, or only the C-terminal tail of BMPR-II. Proteins
which formed complexes with these BMPR-II constructs were analyzed by

two-dimensional gel electrophoresis (2-D GE), and specifically
interacting proteins were identified by matrix-assisted laser
desorption/ionization-time of flight-mass spectrometry (MALDI-TOF-MS). We
identified 33 interacting proteins; 11 proteins interacted with the
C-terminal tail of BMPR-II, 4 with full-length BMPR-II, and 18 with a
short form of the receptor with a deleted tail. Fourteen proteins have
assigned functions in various signalling processes, suggesting links of
BMP signalling to regulation of MAP kinase pathway, apoptosis,
transcription, PKCbeta, and PKA. Five of the identified proteins are
components of the cytoskeleton, and four are enzymes involved in
metabolism, e.g., processing of estrogens or lipids. We confirmed
interaction of PKCbeta and *CtBP* with BMPR-II using immunodetection. We
showed that the C-terminal tail of BMPR-II provides binding sites for a
number of regulatory proteins that may initiate *Smad*-independent
signalling.
? t s4/abs
>>>"ABS" is not a valid format name in file(s): 5, 10, 34, 42, 50, 65, 71,
    73, 91, 94, 143-144, 155-157, 159, 198, 266, 304, 340, 344, 348-349,
    357-358, 370, 434, 444, 447, 467
? t s4/3,ab/all
>>>No matching display code(s) found in file(s): 65, 198, 304, 447

4/3,AB/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0014936627   BIOSIS NO.: 200400307384
Proteins associated with type II bone morphogenetic protein receptor
   (BMPR-II) and identified by two-dimensional gel electrophoresis and mass
   spectrometry
AUTHOR: Hassel Sylke; Eichner Annegret; Yakymovych Mariya; Hellman Ulf;
   Knaus Petra; Souchelnytskyi Serhiy (Reprint)
AUTHOR ADDRESS: BMC, Ludwig Inst Canc Res, Box 595,Husargatan 3, SE-75124,
   Uppsala, Sweden**Sweden
AUTHOR E-MAIL ADDRESS: serhiy.souchelnytskyi@licr.uu.se
JOURNAL: Proteomics 4 (5): p1346-1358 May 2004 2004
MEDIUM: print
ISSN: 1615-9853 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Bone morphogenetic proteins (BMP) are polypeptide growth factors
   that regulate cell differentiation and proliferation. BMPs bind to type I
   and type II serine/threonine kinase receptors to initiate intracellular
   signalling. BMPR-II is the type II receptor, its mutations lead to
   hereditary pulmonary hypertension, and knockout of Bmpr-II results in
   early embryonic lethality. To identify novel interacting proteins and
   explore signalling pathways that can be initiated by BMPR-II, we
   performed glutathione-S-transferase (GST) pull-down assays with BMPR-II
   protein constructs fused to GST and extracts of mouse myoblast C2C12
   cells. We generated three constructs which contain different parts of the
   cytoplasmic region of BMPR-II: full-length cytoplasmic part of BMPR-II,
   only the kinase domain, or only the C-terminal tail of BMPR-II. Proteins
   which formed complexes with these BMPR-II constructs were analyzed by
   two-dimensional gel electrophoresis (2-D GE), and specifically
   interacting proteins were identified by matrix-assisted laser
   desorption/ionization-time of flight-mass spectrometry (MALDI-TOF-MS). We
   identified 33 interacting proteins; 11 proteins interacted with the

C-terminal tail of BMPR-II, 4 with full-length BMPR-II, and 18 with a
short form of the receptor with a deleted tail. Fourteen proteins have
assigned functions in various signalling processes, suggesting links of
BMP signalling to regulation of MAP kinase pathway, apoptosis,
transcription, PKCbeta, and PKA. Five of the identified proteins are
components of the cytoskeleton, and four are enzymes involved in
metabolism, e.g., processing of estrogens or lipids. We confirmed
interaction of PKCbeta and *CtBP* with BMPR-II using immunodetection. We
showed that the C-terminal tail of BMPR-II provides binding sites for a
number of regulatory proteins that may initiate *Smad*-independent
signalling.

4/3,AB/2   (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0014652262   BIOSIS NO.: 200400023019
**Smad6 recruits transcription corepressor *CtBP* to repress bone
  morphogenetic protein-induced transcription.**
AUTHOR: Lin Xia (Reprint); Liang Yao-Yun; Sun Baohua; Liang Min; Shi
  Yujiang; Brunicardi F Charles; Shi Yang; Feng Xin-Hua
AUTHOR ADDRESS: Department of Surgery, Baylor College of Medicine, One
  Baylor Plaza, Room 131D, Houston, TX, 77030, USA**USA
AUTHOR E-MAIL ADDRESS: xialin@bcm.tmc.edu; xfeng@bcm.tmc.edu

ABSTRACT: Smad6 and Smad7 are inhibitory Smads induced by transforming
  growth factor beta-*Smad* signal transduction pathways in a
  negative-feedback mechanism. Previously it has been thought that
  inhibitory Smads bind to the type I receptor and block the
  phosphorylation of receptor-activated Smads, thereby inhibiting the
  initiation of *Smad* signaling. Conversely, few studies have suggested
  the possible nuclear functions of inhibitory Smads. Here, we present
  compelling evidence demonstrating that Smad6 repressed bone morphogenetic
  protein-induced Id1 transcription through recruiting transcriptional
  corepressor C-terminal binding protein (*CtBP*). A consensus *CtBP*
  -binding motif, PLDLS, was identified in the linker region of Smad6. Our
  findings show that mutation in the motif abolished the Smad6 binding to
  CtBP and subsequently its repressor activity of transcription. We
  conclude that the nuclear functions and physical interaction of Smad6 and
  CtBP provide a novel mechanism for the transcriptional regulation by
  inhibitory Smads.

4/3,AB/3   (Item 3 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0014447731   BIOSIS NO.: 200300406450
**Interaction between *Smad*-interacting protein-1 and the corepressor
  C-terminal binding protein is dispensable for transcriptional repression
  of E-cadherin.**

AUTHOR: van Grunsven Leo A; Michiels Christine; Van de Putte Tom; Nelles
    Luc; Wuytens Gunther; Verschueren Kristin (Reprint); Huylebroeck Danny
AUTHOR ADDRESS: Dept. of Developmental Biology, Laboratory of Molecular
    Biology (Celgen), Flanders Interuniversity Institute for Biotechnology
    (VIB), University of Leuven, Herestraat 49, VIB7, Campus Gasthuisberg
    (Bldg. O and N), B-3000, Leuven, Belgium**Belgium
AUTHOR E-MAIL ADDRESS: Kristin.Verschueren@med.kuleuven.ac.be

ABSTRACT: deltaEF1 and SIP1 (or Zfhx1a and Zfhx1b, respectively) are the
    only known members of the vertebrate Zfh1 family of homeodomain/zinc
    finger-containing proteins. Similar to other transcription factors, both
    Smad-interacting protein-1 (SIP1) and deltaEF1 are capable of
    repressing E-cadherin transcription through binding to the E2 boxes
    located in its promoter. In the case of deltaEF1, this repression has
    been proposed to occur via interaction with the corepressor C-terminal
    binding protein (*CtBP*). In this study, we show by coimmunoprecipitation
    that SIP1 and *CtBP* interact in vivo and that an isolated *CtBP*-binding
    SIP1 fragment depends on *CtBP* for transcriptional repression. However,
    and most importantly, full-length SIP1 and deltaEF1 proteins do not
    depend on their interaction with *CtBP* to repress transcription from the
    E-cadherin promoter. Furthermore, in E-cadherin-positive kidney
    epithelial cells, the conditional synthesis of mutant SIP1 that cannot
    bind to *CtBP* abrogates endogenous E-cadherin expression in a similar
    way as wild-type SIP1. Our results indicate that full-length SIP1 can
    repress E-cadherin in a *CtBP*-independent manner.

4/3,AB/4   (Item 4 from file: 5)

0014354932   BIOSIS NO.: 200300312421
**Regulation of *Smad* signaling through a differential recruitment of
    coactivators and corepressors by ZEB proteins.**
AUTHOR: Postigo Antonio A (Reprint); Depp Jennifer L; Taylor Jennifer J;
    Kroll Kristen L
AUTHOR ADDRESS: Division of Molecular Oncology, Department of Internal
    Medicine, Washington University School of Medicine, Saint Louis, MO,
    63110, USA**USA
AUTHOR E-MAIL ADDRESS: apostigo@im.wustl.edu

ABSTRACT: Balancing signals derived from the TGFbeta family is crucial for
    regulating cell proliferation and differentiation, and in establishing
    the embryonic axis during development. TGFbeta/BMP signaling leads to the
    activation and nuclear translocation of *Smad* proteins, which activate

transcription of specific target genes by recruiting P/CAF and p300. The
two members of the ZEB family of zinc finger factors (ZEB-1/deltaEF1 and
ZEB-2/SIP1) regulate TGFbeta/BMP signaling in opposite ways:
ZEB-1/deltaEF1 synergizes with *Smad*-mediated transcriptional
activation, while ZEB-2/SIP1 represses it. Here we report that these
antagonistic effects by the ZEB proteins arise from the differential
recruitment of transcriptional coactivators (p300 and P/CAF) and
,corepressors (*CtBP*) to the Smads. Thus, while ZEB-1/deltaEF1 binds to
p300 and promotes the formation of a p300-*Smad* transcriptional complex,
ZEB-2/SIP1 acts as a repressor by recruiting *CtBP*. This model of
regulation by ZEB proteins also functions in vivo, where they have
opposing effects on the regulation of TGFbeta family-dependent genes
during Xenopus development.

4/3,AB/5   (Item 5 from file: 5)

Oncogenic mechanisms of Evi-1 protein
AUTHOR: Hirai Hisamaru (Reprint); Izutsu Koji; Kurokawa Mineo; Mitani
  Kinuko
AUTHOR ADDRESS: Department of Hematology and Oncology, Graduate School of
  Medicine, University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo, 113-8655,
  Japan**Japan

ABSTRACT: Although Evi-1 is thought to promote growth or block
  differentiation in some cell types, its biological functions have not
  been elucidated. To explore the mechanisms underlying Evi-1-induced
  oncogenesis, we investigated whether Evi-1 affects the signaling of
  transforming growth factor beta (TGF-beta), which inhibits proliferation
  of a wide range of cell types and is one of the most studied growth
  regulatory factors. We demonstrated that Evi-1 represses TGF-beta
  signaling and antagonizes its growth-inhibitory effects. Two separate
  regions of Evi-1 are responsible for this repression, one of which is the
  first zinc-finger domain. Through this domain, Evi-1 physically interacts
  with Smad3, an intracellular mediator of TGF-beta signaling, thereby
  suppressing the transcriptional activity of Smad3. These results define a
  novel function of Evi-1 as a repressor of signaling components of
  TGF-beta. We also demonstrated that Evi-1 represses *Smad*-induced
  transcriptional activation by recruiting *CtBP* as a corepressor. Evi-1
  associates with CtBP1 through one of the *CtBP*-binding consensus motifs
  within the region from amino acid 544 to 607, and this association is
  required for the efficient inhibition of TGF-beta signaling. A specific
  histone deacetylase (HDAc) inhibitor, trichostatin A (TSA), alleviates
  Evi-1-mediated repression of TGF-beta signaling, suggesting that HDAc is
  involved in transcriptional repression by Evi-1. This identifies a novel
  function of Evi-1 as a member of corepressor complexes and suggests that
  aberrant recruitment of corepressors is one of the mechanisms involved in
  Evi-1-induced leukemogenesis. These results indicate that specific HDAc
  inhibitors may be useful in the treatment of Evi-1-induced neoplastic

tumors, including myeloid leukemias.

4/3,AB/6   (Item 6 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0013129631   BIOSIS NO.: 200100301470
The corepressor *CTBP* is involved in Evi-1 mediated repression of TGF-beta signaling
AUTHOR: Izutsu Koji (Reprint); Kurokawa Mineo (Reprint); Imai Yoichi
  (Reprint); Mitani Kinuko (Reprint); Hirai Hisamaru (Reprint)
AUTHOR ADDRESS: Department of Hematology and Oncology, Graduate School of
  Medicine, University of Tokyo, Tokyo, Japan**Japan

ABSTRACT: Evi-1 is a zinc finger nuclear protein whose inappropriate
  expression leads to leukemic transformation of hematopoietic cells in
  mice and humans. Evi-1 is shown to be highly expressed in human myeloid
  leukemias and myelodysplastic syndromes by chromosomal rearrangements
  involving 3q26. It is also aberrantly expressed as a fusion transcript
  with AML1 (AML1/Evi-1), which leads to blastic transformation in patients
  with chronic myelogenous leukemia. We previously showed that Evi-1 and
  AML1/Evi-1 block the antiproliferative effect of TGF-beta. They represses
  TGF-beta signaling by direct interaction with Smad3 through their first
  zinc finger motif. Here, we demonstrate that Evi-1 represses *Smad*
  -induced transcription by recruiting *CtBP* as a corepressor. *CtBP* was
  originally identified as a protein which interacts with C-terminal region
  of adenoviral oncoprotein E1A. *CtBP* is ubiquitously expressed including
  hematopoietic cells, and has been shown to act as a corepressor of
  certain transcriptional repressors, such as BKLF, FOG, and TCF. We show
  that Evi-1 directly associates with CtBP1 through one of the consensus
  binding motifs, and this association is required for efficient inhibition
  of TGF-beta signaling. A specific inhibitor for histone deacetylase
  (HDAc) alleviates Evi-1-mediated repression of TGF-beta signaling,
  suggesting that HDAc is involved in the transcriptional repression by
  Evi-1. This identifies a novel function of Evi-1 as a member of
  corepressor complexes and suggests that aberrant recruitment of
  corepressors is one of the mechanisms for Evi-1-induced leukemogenesis.

4/3,AB/7   (Item 7 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0013072727   BIOSIS NO.: 200100244566
The corepressor *CtBP* interacts with Evi-1 to repress transforming growth factor beta signaling
AUTHOR: Izutsu Koji; Kurokawa Mineo; Imai Yoichi; Maki Kazuhiro; Mitani
  Kinuko; Hirai Hisamaru (Reprint)
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ABSTRACT: Evi-1 is a zinc finger nuclear protein whose inappropriate expression leads to leukemic transformation of hematopoietic cells in mice and humans. This was previously shown to block the antiproliferative effect of transforming growth factor beta (TGF-beta). Evi-1 represses TGF-beta signaling by direct interaction with Smad3 through its first zinc finger motif. Here, it is demonstrated that Evi-1 represses *Smad* -induced transcription by recruiting C-terminal binding protein (*CtBP*) as a corepressor. Evi-1 associates with CtBP1 through one of the consensus binding motifs, and this association is required for efficient inhibition of TGF-beta signaling. A specific inhibitor for histone deacetylase (HDAc) alleviates Evi-1-mediated repression of TGF-beta signaling, suggesting that HDAc is involved in the transcriptional repression by Evi-1. This identifies a novel function of Evi-1 as a member of corepressor complexes and suggests that aberrant recruitment of corepressors is one of the mechanisms for Evi-1-induced leukemogenesis.

4/3,AB/8   (Item 8 from file: 5)

0012889338   BIOSIS NO.: 200100061177
The interaction of the carboxyl terminus-binding protein with the *Smad* corepressor TGIF is disrupted by a holoprosencephaly mutation in TGIF
AUTHOR: Melhuish Tiffany A; Wotton David (Reprint)
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ABSTRACT: The homeodomain protein TGIF represses transcription in part by recruiting histone deacetylases. TGIF binds directly to DNA to repress transcription or interacts with TGF-beta-activated Smads, thereby repressing genes normally activated by TGF-beta. Loss of function mutations in TGIF result in holoprosencephaly (HPE) in humans. One HPE mutation in TGIF results in a single amino acid substitution in a conserved PLDLS motif within the amino-terminal repression domain. We demonstrate that TGIF interacts with the corepressor carboxyl terminus-binding protein (*CtBP*) via this motif. *CtBP*, which was first identified by its ability to bind the adenovirus E1A protein, interacts both with gene-specific transcriptional repressors and with a subset of polycomb proteins. Efficient repression of TGF-beta-activated gene responses by TGIF is dependent on interaction with *CtBP*, and we show that TGIF is able to recruit *CtBP* to a TGF-beta-activated *Smad* complex. Disruption of the PLDLS motif in TGIF abolishes the interaction

of *CtBP* with TGIF and compromises the ability of TGIF to repress
transcription. Thus, at least one HPE mutation in TGIF appears to prevent
CtBP-dependent transcriptional repression by TGIF, suggesting an
important developmental role for the recruitment of *CtBP* by TGIF.

4/3,AB/9   (Item 1 from file: 340)

**COMPOSITIONS AND METHODS FOR NEGATIVE REGULATION OF TGF-BETA PATHWAYS;
IDENTIFYING COMPOUNDS THAT DIRECTLY INTERACT WITH *SMAD* PROTEINS, OR WITH
SMAD CO-REPRESSORS, TO PREVENT PROTEIN-PROTEIN OR PROTEIN-DNA
INTERACTIONS REQUIRED FOR TRANSCRIPTIONAL REPRESSION IN RESPONSE TO TGF,
ACTIVIN**
Inventors: Laughon Allen S (US)
Assignee: Unassigned Or Assigned To Individual
Assignee Code: 68000

Abstract: Methods for screening for compounds that are negative regulators
of TGF- beta -regulated gene expression in mammalian cells are provided,
including compositions identified therefrom.

4/3,AB/10   **(Item 1 from file: 73)**

  TGIF2 Interacts with Histone Deacetylase I and Represses Transcription
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  TG-interacting factor (TGIF) is a transcriptional repressor, which
represses transcription by binding directly to DNA or interacts with
transforming growth factor alpha (TGFbeta)-activated Smads, thereby
repressing TGFbeta-responsive gene expression. Mutation of TGIF in humans
causes holoprosencephaly, a severe genetic disorder affecting craniofacial
development. Searching human expressed sequence tag data bases revealed the
presence of clones encoding a TGIF-related protein (TGIF2), which contains
two regions of high sequence identity with TGIF. Here we show that, like
TGIF, TGIF2 recruits histone deacetylase, but in contrast to TGIF, is
unable to interact with the corepressor *CtBP*. TGIF2 and TGIF have very
similar DNA-binding homeodomains, and TGIF2 represses transcription when

bound to DNA via a TGIF binding site. TGIF2 interacts with TGFbeta-activated Smads and represses TGFbeta-responsive transcription. TGIF2 appears to be a context-independent transcriptional repressor, which can perform similar functions to TGIF and may play a role in processes, which, when disrupted by mutations in TGIF, cause holoprosencephaly.

4/3,AB/11   (Item 1 from file: 155)
  Repression   of   bone   morphogenetic   protein   and   activin-inducible transcription by Evi-1.
  Alliston Tamara; Ko Tien C; Cao Yanna; Liang Yao-Yun; Feng Xin-Hua; Chang Chenbei; Derynck Rik
  Department of Cell and Tissue Biology, University of California at San Francisco, San Francisco, California 94143-0512, USA.
  Smads, key effectors of transforming growth factor (TGF)-beta, activin, and bone morphogenetic protein (BMP) signaling, regulate gene expression and interact with coactivators and corepressors that modulate *Smad* activity. The corepressor Evi-1 exerts its oncogenic effects by repressing TGF-beta/Smad3-mediated transcription, thereby blocking TGF-beta-induced growth arrest. Because Evi-1 interacts with the highly conserved MH2 domain of Smad3, we investigated the physical and functional interaction of Evi-1 with Smad1 and Smad2, downstream targets of BMP and activin signaling, respectively. Evi-1 interacted with and repressed the receptor-activated transcription through Smad1 and Smad2, similarly to Smad3. In addition, Evi-1 repressed BMP/Smad1- and activin/Smad2-mediated induction of endogenous Xenopus gene expression, suggesting a role of repression of BMP and activin signals by Evi-1 in vertebrate embryogenesis. Evi-1 also repressed the induction of endogenous Smad7 expression by TGF-beta family ligands. In the course of these studies, we observed Evi-1 repression of *Smad* transactivation even when *Smad* binding to DNA was kept constant. We therefore explored the mechanism of Evi-1 repression of TGF-beta family-inducible transcription. Evi-1 repression did not result from displacement of *Smad* binding to DNA or to CREB-binding protein but from the recruitment of Evi-1 by Smad3 and CREB-binding protein to DNA. Following TGF-beta stimulation, Evi-1 and the associated corepressor *CtBP* were recruited to the endogenous Smad7 promoter. Evi-1 recruitment to the promoter decreased TGF-beta-induced histone acetylation, coincident with its repression of Smad7 gene expression. In this way, Evi-1 acts as a general *Smad* corepressor to inhibit TGF-beta-, activin-, and BMP-inducible transcription.

4/3,AB/12   (Item 1 from file: 349)

01116723

BINARY PREDICTION TREE MODELING WITH MANY PREDICTORS AND ITS USES IN CLINICAL AND GENOMIC APPLICATIONS

MODELISATION D'UN ARBRE PREVISIONNEL BINAIRE A PLUSIEURS PREDICTEURS, ET SON UTILISATION DANS DES APPLICATIONS CLINIQUES ET GENOMIQUES

English Abstract
  Provided is a statistical analysis method that is a predictive
  statistical tree model. This model first screens genes to reduce noise,
  applies k-means correlation-based clustering, and then uses
  singular-value decompositions to extract the single dominant factor
  (principal component) from each cluster. This generates a statistically
  significant number of cluster-derived singular factors, that we refer to
  as metagenes, which characterize multiple patterns of expression of the
  genes across samples. The strategy aims to extract multiple such patterns
  while reducing dimension and smoothing out gene[specific noise through
  the aggregation within clusters. Formal predictive analysis then uses
  these metagenes in a Bayesian classification tree analysis. This
  generates multiple recursive partitions of the sample into subgroups
  ("leaves" of the tree), and associates Bayesian predictive probabilities
  of outcomes with each subgroup. Overall predictions for an individual

sample are then generated by averaging predictions, with appropriate
weights, across many such tree models. The model includes the use of
iterative out-of-sample cross-validation predictions to perform refitting
of the model, and mirrors the real-world prognostic context where
prediction of new cases as they arise is the major goal.

French Abstract
L'invention porte sur une analyse statistique sous forme de modele
statistique previsionnel d'arborescence resolvant plusieurs problemes
observes dans des modeles statistiques anterieurs et des analyses de
regression, tout en offrant une precision et des capacites
previsionnelles ameliorees. Bien que le modele de l'invention serve
principalement a pronostiquer les maladie d'individus, il peut egalement
etre utilise dans une variete d'applications dont: la prevision des
stades de maladies ou de la susceptibilite d'y arriver, tout autre etat
biologique d'interet, et d'autres etats non biologiques d'interet. Le
modele de l'invention crible d'abord les genes pour reduire le bruit,
applique k moyens d'agglutination a base de correlation a un grand nombre
d'utilisations, puis procede a une decomposition en valeurs singulieres
pour extraire le facteur dominant unique (composant principal) de chacun
des amas. Cela cree un nombre statistiquement significatif de facteurs
singuliers, dits metagenes caracterisant de multiples schema d'expression
des genes dans les echantillons. La strategie vise a extraire nombre de
ces schemas tout en reduisant les dimensions et lissant le bruit
specifique des genes en les agregeant en amas. L'analyse predictive
formelle utilise alors ces metagenes pour une analyse par arbre Bayesien
de classification. Cela cree de multiples separations recursives de
l'echantillon en sous-groupes (les feuilles de l'arbre de classification)
et les probabilites previsionnelles associees Bayesiennes des resultats
pour chaque sous-groupe. Les previsions generales relatives a un
echantillon individuel sont alors etablies par moyennage avec des poids
appropries en utilisant plusieurs de ces modeles d'arborescence. Le
modele de l'invention utilise des pronostics iteratifs hors
echantillonnage et des pronostics a validation croisee, laissant chaque
echantillon un par un hors de l'ensemble de donnees, rajustant le modele
a partir des echantillons restants et l'utilisant pour pronostiquer les
cas a ecarter. Cela verifie ainsi rigoureusement les valeurs
previsionnelles d'un modele et reflete le contexte des pronostics en
temps reel alors que les previsions sur les nouveaux cas se presentant
est l'objectif majeur.

4/3,AB/13   (Item 2 from file: 349)
DIALOG(R)File 349:PCT FULLTEXT
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01094160
**EXPANSION OF CELLS USING THROMBOPOIETIN AND ANTI-TRANSFORMING GROWTH
FACTOR-BETA**
**DEVELOPPEMENT DE CELLULES UTILISANT LA THROMBOPOIETINE ET UN
ANTI-FACTEUR-BETA DE CROISSANCE TRANSFORMANT**
Patent Applicant/Assignee:
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states except: US)
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Patent and Priority Information (Country, Number, Date):
  Patent: WO 200414939 A2-A3 20040219 (WO 0414939)
  Application: WO 2003US24929 20030807 (PCT/WO US03024929)
  Priority Application: US 2002213957 20020807
Parent Application/Grant:
  Related by Continuation to: US 2002213957 20020807 (CON)
Designated States:
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  AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ
  EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR
  LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD
  SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
  (EP) AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT RO SE
  SI SK TR
  (OA) BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
  (AP) GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
  (EA) AM AZ BY KG KZ MD RU TJ TM
Publication Language: English
Filing Language: English
Fulltext Word Count: 16936

English Abstract

  The invention features a method for the expansion of hematopoietic stem
  cells using a combination of a thrombopoietin agonist and a transforming
  growth factor- beta blocking agent in the absence of stem cell factor.
  The invention also features a hematopoietic stem cell composition that
  has been expanded using a combination of a thrombopoietin agonist and a
  transforming growth factor-beta blocking agent in the absence of stem
  cell factor, as well as methods of using an expanded hematopoietic stem
  cell composition to restore or supplement an immune system and/or blood
  forming system compromised by, for example, radiation or chemotherapy.

French Abstract

  L'invention concerne un procede de developpement de cellules souches
  hematopoietiques utilisant une combinaison d'un agoniste de
  thrombopoietine et d'un agent bloquant le facteur beta de croissance
  transformant en l'absence de facteur de cellules souches. L'invention
  concerne egalement une composition de cellules souches hematopoietiques
  ayant ete developpees au moyen d'une combinaison d'un agoniste de
  thrombopoietine et d'un agent bloquant le facteur beta de facteur
  transformant en l'absence de facteur de cellules souches ainsi que des
  procedes d'utilisation d'une composition de cellules souches
  hematopoietiques developpees pour retablir ou completer un systeme immun
  et/ou un systeme hematopoietique defaillant, par exemple, par rayonnement
  ou chimiotherapie.

4/3,AB/14   (Item 3 from file: 349)
DIALOG(R)File 349:PCT FULLTEXT
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01044797
**RNA INTERFERENCE MEDIATED INHIBITION OF GENE EXPRESSION USING SHORT
    INTERFERING NUCLEIC ACID (SINA)**
**INTERFERENCE ARN PERMETTANT D'INHIBER L'EXPRESSION D'UN GENE A L'AIDE D'UN
    ACIDE NUCLEIQUE INTERFERANT COURT (SINA)**

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Patent and Priority Information (Country, Number, Date):
  Patent: WO 200374654 A2-A3 20030912 (WO 0374654)
  Application: WO 2003US5028 20030220 (PCT/WO US03005028)
  Priority Application: US 2002358580 20020220; US 2002363124 20020311; US
    2002386782 20020606; US 2002406784 20020829; US 2002408378 20020905; US
    2002409293 20020909; US 2003440129 20030115
Parent Application/Grant:
  Related by Continuation to: US 2002358580 20020220 (CON); US 2002363124
    20020311 (CON); US 2002386782 20020606 (CON); US 2002406784 20020829
    (CON); US 2002408378 20020905 (CON); US 2002409293 20020909 (CON); US
    2003440129 20030115 (CON)
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  AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ
  EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR
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  SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
  (EP) AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT SE SI
  SK TR
  (OA) BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
  (AP) GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
  (EA) AM AZ BY KG KZ MD RU TJ TM
Publication Language: English
Filing Language: English
Fulltext Word Count: 262755

English Abstract
  The present invention concerns methods and reagents useful in modulating
  gene expression in a variety of applications, including use in
  therapeutic, diagnostic, target validation, and genomic discovery
  applications. Specifically, the invention relates to small nucleic acid
  molecules, such as short interfering nucleic acid (siNA), short
  interfering RNA (siRNA), double-stranded RNA (dsRNA), micro-RNA (miRNA),
  and short hairpin RNA (shRNA) molecules capable of mediating RNA
  interference (RNAi) against target nucleic acid sequences. The small
  nucleic acid molecules are useful in the treatment of any disease or

condition that responds to modulation of gene expression or activity in a cell, tissue, or organism.

French Abstract

L'invention concerne des methodes et des reactifs utilises pour moduler une expression genique dans une variete d'applications, notamment dans des applications therapeutiques, diagnostiques, de validation de cible et de decouverte genomique. L'invention concerne, plus specifiquement, de petites molecules d'acide nucleique, tel qu'un acide nucleique d'interference court (INA), un ANR d'interference court (siRNA), un ARN double brins (dsRNA), un micro-ARN (miRNA) et des molecules d'ARN court en epingle a cheveux (shRNA) capables d'induire une mediation d'interference ARN (RNAi) par rapport a des sequences cibles d'acide . nucleique. On utilise ces petites molecules d'acide nucleiques pour traiter une maladie ou un etat quelconque repondant a la modulation de l'expression d'un gene ou d'une activite dans une cellule, un tissu, ou un organisme.

4/3,AB/15   (Item 4 from file: 349)
DIALOG(R)File 349:PCT FULLTEXT

00942700
COMPOSITIONS AND METHODS FOR NEGATIVE REGULATION OF TGF-BETA PATHWAYS
COMPOSITIONS   ET   PROCEDES POUR LA REGULATION NEGATIVE DES VOIES DE FACTEUR DE CROISSANCE TRANSFORMANT BETA
Patent Applicant/Assignee:
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Inventor(s):
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Legal Representative:
  LICATA Jane Massey (et al) (agent), Licata & Tyrrell P.C., 66 E. Main Street, Marlton, NJ 08053, US,
Patent and Priority Information (Country, Number, Date):
  Patent: WO 200276466 A1 20021003 (WO 0276466)
  Application: WO 2002US8133 20020315 (PCT/WO US0208133)
  Priority Application: US 2001810385 20010316
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  AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ
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  LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI
  SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW
  (EP) AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
  (OA) BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
  (AP) GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
  (EA) AM AZ BY KG KZ MD RU TJ TM
Publication Language: English
Filing Language: English
Fulltext Word Count: 4842

English Abstract
  Methods for screening for compounds that are negative regulators of TGF-beta-regulated gene expression in mammalian cells are provided, including compositions identified therefrom.

French Abstract
   La presente invention concerne des procedes de criblage de composes qui
   sont des regulateurs negatifs de l'expression genetique regulee de
   TGF-beta dans des cellules mammaliennes, ainsi que des compositions qui
   sont identifiees a partir de ceux-ci. FIG. 1 : A beta-GALACTOSIDASE B 5
   NANOGRAMMES DE MAD/20 NANOGRAMMES DE MEDEA C 0 NANOGRAMMES DE MEDEA, 0
   NANOGRAMMES DE MAD D NANOGRAMMES DE LA PROTEINE dCtB

4/3,AB/16   (Item 5 from file: 349)
DIALOG(R)File 349:PCT FULLTEXT
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00739915
GENE EXPRESSION IN BLADDER TUMORS
EXPRESSION GENIQUE DANS LES TUMEURS DE LA VESSIE
Patent Applicant/Inventor:
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Legal Representative:
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      Hamburg, DE,
Patent and Priority Information (Country, Number, Date):
   Patent: WO 200052204 A2-A3 20000908 (WO 0052204)
   Application: WO 2000IB367 20000222 (PCT/WO IB0000367)
   Priority Application: US 99121124 19990222
Parent Application/Grant:
   Related by Continuation to: US 99121124 19990222 (CON)
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   AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB
   GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA
   MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA
   UG US UZ VN YU ZA ZW
   (EP) AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
   (OA) BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
   (AP) GH GM KE LS MW SD SL SZ TZ UG ZW
   (EA) AM AZ BY KG KZ MD RU TJ TM
Publication Language: English
Filing Language: English
Fulltext Word Count: 161096

English Abstract
   Methods for analyzing tumor cells, particularly bladder tumor cells
   employ gene expression analysis of samples. Gene expression patterns are
   formed and compared to reference patterns. Alternatively gene expression
   patterns are manipulated to exclude genes which are expressed in
   contaminating cell populations. Another alternative employs subtraction
   of the expression of genes which are expressed in contaminating cell
   types. These methods provide improved accuracy as well as alternative
   basis for analysis from diagnostic an prognostic tools currently
   available.

French Abstract
   L'invention concerne des procedes d'analyse des cellules cancereuses,
   particulierement des cellules cancereuses de la vessie recourant a
   l'analyse genique d'echantillons. Les modeles d'expression genique sont
   formes et compares a des modeles de reference. Selon une variante, les

modeles d'expression genique sont manipules pour exclure les genes qui
sont exprimes dans des populations de cellules contaminantes. Selon une
autre variante, on utilise la soustraction de l'expression des genes qui
sont exprimes dans des types de cellules contaminantes. Ces procedes
assurent une plus grande precision et servent de base pour l'analyse a
partir d'outils de diagnostic et de pronostic disponibles sur le marche.

4/3,AB/17   (Item 1 from file: 34)

11659340   Genuine Article#: 680BU   Number of References: 66
**Title: Opposing functions of ZEB proteins in the regulation of the TGF
    beta/BMP signaling pathway** (ABSTRACT AVAILABLE)
Author(s): Postigo AA   (REPRINT)
Corporate Source: Washington Univ,Sch Med, Dept Internal Med, Div Mol
    Oncol,St Louis//MO/63110 (REPRINT); Washington Univ,Sch Med, Dept
    Internal Med, Div Mol Oncol,St Louis//MO/63110
Language: English   Document Type: ARTICLE
Abstract: Binding of TGFbeta/BMP factors to their receptors leads to
    translocation of *Smad* proteins to the nucleus where they activate
    transcription of target genes. The two-handed zinc finger proteins
    encoded by Zfhx1a and Zfhx1b, ZEB-1/deltaEF1 and ZEB-2/SIP1,
    respectively, regulate gene expression and differentiation programs in
    a number of tissues. Here I demonstrate that ZEB proteins are also
    crucial regulators of TGFbeta/BMP signaling with opposing effects on
    this pathway. Both ZEB proteins bind to Smads, but while ZEB-1/deltaEF1
    synergizes with *Smad* proteins to activate transcription, promote
    osteoblastic differentiation and induce cell growth arrest, the highly
    related ZEB-2/SIP1 protein has the opposite effect. Finally, the
    ability of TGFbeta to mediate transcription of TGFbeta-dependent genes
    and induce growth arrest depends on the presence of endogenous
    ZEB-1/deltaEF1 protein.

4/3,AB/18   (Item 2 from file: 34)

10819811   Genuine Article#: 571XC   Number of References: 52
**Title: Overlapping and unique roles for C-terminal binding protein 1
    (CtBP1) and CtBP2 during mouse development.** (ABSTRACT AVAILABLE)
Author(s): Hildebrand JD (REPRINT) ; Soriano P
Corporate Source: Univ Pittsburgh,Dept Biol Sci,5th & Ruskin
    Ave/Pittsburgh//PA/15260 (REPRINT); Univ Pittsburgh,Dept Biol
    Sci,Pittsburgh//PA/15260; Fred Hutchinson Canc Res Ctr,Program Dev
    Biol,Seattle//WA/98108; Fred Hutchinson Canc Res Ctr,Div Basic
    Sci,Seattle//WA/98108
Language: English   Document Type: ARTICLE
Abstract: The C-terminal binding protein (*CtBP*) family of proteins has
    been linked to multiple biological processes through their association

with numerous transcription factors. We generated mice harboring
mutations in both Ctbp1 and Ctbp2 to address the in vivo function of
CtBPs during vertebrate development. Ctbp1 mutant mice are small but
viable and fertile, whereas Ctbp2-null mice show defects in axial
patterning and die by E10.5 due to aberrant extraembryonic development.
Mice harboring various combinations of Ctbp1 and Ctbp2 mutant alleles
exhibit dosage-sensitive defects in a wide range of developmental
processes. The strong genetic interaction, as well as transcription
assays with *CtBP*-deficient cells, indicates that CtBPs have
overlapping roles in regulating gene expression. We suggest that the
observed phenotypes reflect the large number of transcription factors
whose activities are compromised in the absence of *CtBP*.

4/3,AB/19   (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.
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0323067 DBR Accession No.: 2003-24207   PATENT
Identifying compounds that interact with *Smad* protein (co-repressor),
useful for treating diseases involving negative regulation of
transforming growth factor-beta e.g. cancer and autoimmune disease -
involving vector-mediated gene transfer and expression in host cell for
use in drug screening
AUTHOR: LAUGHON A S
PATENT ASSIGNEE: LAUGHON A S 2002
PATENT NUMBER: US 20020137662 PATENT DATE: 20020926 WPI ACCESSION NO.:
    2003-657220 (200362)
PRIORITY APPLIC. NO.: US 810385 APPLIC. DATE: 20010316
NATIONAL APPLIC. NO.: US 810385 APPLIC. DATE: 20010316
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Identifying compounds that directly
    interact with a *Smad* protein or a *Smad* protein co-repressor to
    prevent protein-protein or protein-DNA interactions required for
    repression of transcription induced by transforming growth factor
    (TGF)-beta, activin or bone morphogenetic protein (BMP) signaling in
    cells, is new. DETAILED DESCRIPTION - Identifying compounds that
    directly interact with a *Smad* protein or a *Smad* protein
    co-repressor to prevent protein-protein or protein-DNA interactions
    required for repression of transcription induced by transforming growth
    factor (TGF)-beta, activin or bone morphogenetic protein (BMP)
    signaling in cells comprising: (a) determining a first level of
    transcription detected in cells in the presence of a *Smad* protein and
    a *CtBP* (undefined) protein before addition of a test compound; (b)
    contacting the cells with the test compound; and (c) determining a
    second level of transcription detected in cells in the presence of a
    Smad protein and a *CtBP* protein after addition of the test
    compound, where a decrease in the level of repression of transcription
    induced by the presence of the *Smad* protein and the *CtBP* protein is
    indicative of the ability of the test compound to interfere with
    transcriptional repression and to prevent repression of transcription
    that is produced by a TGF-beta, activin, or BMP signal in cells.
    INDEPENDENT CLAIMS are also included for the following: (1) a
    composition identified by the method; and (2) identifying a candidate
    gene that is directly and negatively regulated by TGF-beta signaling
    pathways through a *CtBP* protein comprising: (a) determining a first
    level of TGF-beta-regulated target gene expression in the presence of
    CtBP ; (b) determining a second level of TGF-beta-regulated target
    gene expression in the absence of the *CtBP* protein; and (c) comparing

the first level of expression with the second level of expression,
where dependence of TGF-beta-regulated gene expression on the presence
of the *CtBP* protein is indicative of the ability of the candidate
gene to be directly and negatively regulated by *CtBP* working in
conjunction with the *Smad* protein. BIOTECHNOLOGY - Preferred Method:
Transcription levels both before and after addition of the test
compound are detected in cells in the presence of a *Smad* protein, a
CtBP protein, and a co-repressor protein such as Evi-1, TGIF
(undefined), SIP1 (undefined), and Schnurri. The *Smad* protein is
Drosophila Mad or Medea. The *CtBP* protein is dCtBP (preferred), CtBP2
or any homolog of *CtBP*. ACTIVITY - Cytostatic; Immunosuppressive.
MECHANISM OF ACTION - *CtBP* inhibitor; *Smad* inhibitor; Negative
regulator of TGF-beta. No biological data given. USE - The compounds or
genes identified through such assays would be useful in the development
of drugs and therapeutics for treatment of cancer, autoimmune diseases,
and other hereditary diseases that involve negative regulation by
TGF-beta pathways. (7 pages)

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Set	Items	Description
S1	1	LEVEL OF TRANSCRIPTION
S2	1292	CTBP
S3	53	S2 AND SMAD
S4	19	RD (unique items)

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